junctiva and, consequently, chronic red eyes. Cessation of contact lens wear will usually eliminate these signs and symptoms.

There are many other causes of red eye but in most instances the major ones are easy to diagnose simply by taking a careful history and examining the patient, keeping in mind the different parts of the eye and the ocular adnexa which can be involved.

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Glaucoma and Ocular Hypertension

GLAUCOMA IS A complex disease associated with increased intraocular pressure, abnormality of aqueous outflow, cupping of the optic nerve head and loss of visual field. It is generally agreed that the higher the intraocular pressure, the greater the likelihood of visual field loss. Conversely, the lower the intraocular pressure is maintained, the less likely it is that visual field loss will occur or progress.

Historically, statistics on glaucoma have been garnered by evaluation of the intraocular pressures measured with the classical Schiotz corneal indentation tonometer. Using this device, the statistical distribution of normality for intraocular pressure averages 16 mm of mercury ± 2.8 mm of mercury, with over 90 percent of persons in the normal population having intraocular pressures of less than 21 mm of mercury. Another 2.5 percent of normal persons will have pressures of greater than 21 mm of mercury and less than 0.15 percent will have intraocular pressures of greater than 24 mm of mercury. Although patients with glaucoma at times may have normal or high normal intraocular pressures, it is very uncommon for normal patients to have pressures above 24 mm of mercury.

Recently, various tonometric devices involving corneal flattening (applanation) have allowed for refinements in intraocular pressure measurements. The values of intraocular pressure in the same patient with different tonometers may vary 3 to 5 mm of mercury, depending upon the instrument used and whether the patient is examined standing or lying down. Consequently, it becomes essential to know the type of instrument utilized and the postural attitude of the patient in order to know whether he is normotensive or hypertensive. Simply stated, a 22 mm of mercury pressure with one instrument may be equivalent to a 19 mm of mercury intraocular pressure with another device.

In the most common form of glaucoma the disease is slowly developing and slowly progressive. From early laboratory evidence of disease to manifest visual field loss may take years with or without treatment. Glaucoma is an asymmetric disease, affecting the two eyes differently in the same patient. Often the pressure and field loss may vary considerably between the two eyes; or the pressure may be equivalent in the two eyes yet produce greater functional loss in one eye than the other. The concepts of normal or acceptable intraocular pressure as opposed to pathological intraocular pressure are relative ones and depend upon various factors including the patient's age, systemic blood pressure, rate of secretion of aqueous humor, chamber angle anatomy, pressure in the episcleral veins, vascular perfusion of the optic nerve and individual anatomic susceptibility of the particular optic nerve to damage. What may be a tolerable intraocular pressure for one eye may be a pathologic pressure for another.

Whether by a direct effect or by an ischemic mechanism, intraocular pressure is the culprit in a glaucomatous eye. Treatment of glaucoma involves lowering the intraocular pressure by a variety of techniques including drops, pills and surgical operation. Unfortunately, these are not without some risk (diminished visual acuity, myopia, cataracts, retinal detachments and the like). Therefore, a philosophic problem concerning therapy has arisen in the ophthalmologic community. It is a question of whether glaucoma therapy to prevent retinal and optic nerve damage should be used or therapy instituted after such damage has occurred. For cases of moderate or advanced glaucoma, treatment decisions are usually easily made. In borderline cases, decisions are more difficult and must be based on the individual case.

The term ocular hypertension has been introduced to describe persons with elevated intraocular pressure but not known (manifest) ocular damage. The term is somewhat of a misnomer because it includes some patients with high normal

intraocular pressures (21 to 24 mm of mercury) and has even been extended by some to include patients with higher pressures but no field loss or optic nerve damage.

Promotion of the term ocular hypertension has confused some physicians and many patients. It is not a unique entity, nor does it represent a class of patients resistant to abnormal intraocular pressures. Such patients should be suspected of having glaucoma, and frequently become glaucomatous. In clinical studies of persons with "ocular hypertension" the incidence of short-term field loss has ranged from 3.5 percent to 15 percent. Discriminating between ocular hypertension (frequently glaucoma at an early stage) and established chronic simple glaucoma depends upon the status of the optic disc and the visual field. Thorough and periodic examinations are a requisite to proper diagnosis and management. These patients should be followed much more carefully than the general ophthalmologic population.

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Fluorescein Angiography in Ophthalmology

FLUORESCEIN ANGIOGRAPHY has become a fairly common office test for determining the blood flow in the retina and choroid. It has been used to investigate diseases such as macular degeneration and diabetic retinopathy. The ability to determine the cause of fluid in the retina or abnormalities in the retinal vasculature has led to a better understanding of the basic pathophysiology of retinal and choroidal diseases. In turn, the understanding of these entities has led to earlier diagnosis and treatment of these debilitating, ophthalmological diseases.

Fluorescein angiography is done on an outpatient basis. Fluorescein dye (5 to 10 ml) is injected into the antecubital vein as a bolus and serial photographs are taken as the dye passes into the eye from the ophthalmic artery.

A normal fluorescein pattern is seen in Figure 1. As the timed photographs are taken, the advent of leakage from abnormal choriocapillaris areas or retinal vessel breakdowns (or both) become

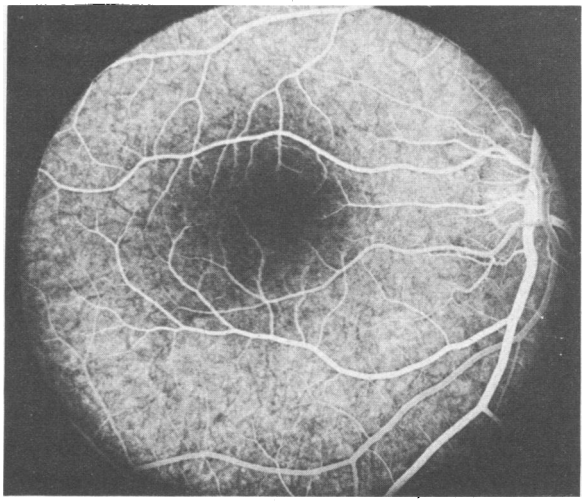

Figure 1.—An angiogram showing a normal fluorescein pattern.

evident. After the choroidal flush becomes apparent, the dye enters the arterial network, which marks the beginning of the arterial phase. The lamellar flow of dye along the edges of the vein marks the beginning of the venous phase and the early venous phase is followed by more uniform filling of the veins. Any abnormal or new retinal vessels as well as old vessels which have undergone deterioration are pinpointed by leakage of the dye through the walls of the abnormal vessels. These leakage spots are pinpointed as bright, white areas of fluorescence which indicate the source of the fluid accumulation in the retina. In many cases, argon laser photocoagulation can be used to seal these areas preventing further deterioration of the retina due to accumulated fluid.

Fluorescein leakage around the optic nerve, which is found in papilledema, is an excellent way of separating cases of papilledema from pseudopapilledema caused by such entities as drusen in the optic nerve head. The test is also useful in determining the presence of ocular tumors. Retinal hemangiomas, metastatic carcinomas and malignant melanomas tend to leak large amounts of the fluorescein dye.

Reactions to the dye can be expected. The patient will have green urine as the dye is excreted by the urinary system. The skin will be slightly pale for 12 hours due to dye absorption. The most frequent complication is nausea, which will occur in about 2 percent of patients at the time the dye is injected and will last from 15 to 30 seconds. In approximately one person in